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A STUDY OF THE LOWER END OF THE ESOPHAGUS.

By Harris P. Mosher, M. D.,
Boston,
and
G. W. McGregor, M. B.,
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A STUDY OF THE LOWER END OF THE ESOPHAGUS.*

By Harris P. Mosher, M. D.,

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For the past two or three years there has been a great revival of interest in cardiospasm. The English laryngologists have been especially active in discussing the condition. This revival of interest is due to the fortunate fact that a number of cases of cardiospasm have come to autopsy, and a fundamental observation resulted from the study of the specimens, namely, that the nerve control of the musculature of the esophageal wall, located between the two chief muscular layers in groups of ganglion cells known as Auerbach's plexus, is diminished or in places abolished, due to pathologic changes which have destroyed the ganglion cells. Stokes was the first to make this observation. Others have confirmed it, notably Kelley and Hurst. This observation is a very welcome addition to our previous scanty knowledge of the microscopic pathology of the esophagus.

Subject.—The title of the paper is "A Study of the Lower End of the Esophagus." The first part deals with the histology of the normal esophagus from infancy to 61 years of age. It deals also with the microscopic findings in my one autopsy specimen from a case of cardiospasm. The second part is clinical, and deals with a series of nine cases of cardiospasm

*Read at the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Toronto, January 21, 1928.

The X-rays illustrating this article were taken by Dr. A. S. Macmillan, roentgenologist of the Massachusetts Eye and Ear Infirmary.

in which the diagnostic barium bougie was used and in which dilatation of the lower end of the esophagus was carried out under sight.

Material—

Fetus, five months	1
Microscopic sections.	
Infants dead at birth	3
Hardened in formalin and anterior wall of esophagus and stomach removed.	
Infants dead at birth	2
Microscopic sections of esophagus.	
Infant, one year	1
Microscopic sections.	
Child, two years	1
Microscopic sections.	
Child, twelve years	1
Microscopic sections.	
Adults, 40-61 years	5
Microscopic sections.	14

These adult specimens included a marked case of arteriosclerosis; a case which died of cardiorenal disease; a case of stricture of the esophagus which died of septic pleurisy and peritonitis after perforation of the esophagus by a bougie; a case of cardiospasm of fifteen years' duration which died of tuberculosis of the lung.

Conclusions.—In the gross and microscopic examination of fourteen specimens of the human esophagus, varying in ages from birth to 61 years, neither the writer nor Dr. McGregor, who has done the microscopic work for this paper—he has examined 1,500 sections—have been able to find an anatomic sphincter at the cardiac end.

Physiologic experiments have shown that the musculature of the lower end of the esophagus or of the upper end of the stomach acts as a sphincter. In the resting state the terminal portion of the esophagus is practically closed. This is due to the presence of deep vertical folds and much elastic tissue. In

the esophagus the elastic tissue is most abundant at the lower end. The natural twist of the terminal portion of the esophagus helps to maintain the closure. The pressure of the left crus from behind and of the liver from the front aid in the same result. In a typical case of cardiospasm the obstruction at the lower end of the esophagus is slight. It is often accompanied by a backward bend of the terminal portion. An instance of this backward bend has been found by the writer at birth. It was associated with an upward swing of the stomach so that its long axis was horizontal. This change in the position of the stomach may be a causative factor in the deformity of the esophagus.

In nine consecutive cases of cardiospasm the diagnostic barium bougie has shown a waistlike constriction of large caliber —19 to 20 mm.—at the terminal portion of the esophagus above the level of the crura. In all but two of the cases the narrowing dilated under light pressure (three or four pounds) until the esophagus reached its normal width—31 mm.

The waistlike constriction takes the form of the lung tips and is most pronounced when they are inflated.

The Sippey rubber dilator, painted with stripes of barium and rubber cement, enables the operator to dilate the lower end of esophagus under sight.

The observation made by Stokes that in cases of cardiospasm the ganglion cells of Auerbach's plexus are much diminished and in places practically wanting was found to hold in the one case of the writer's which came to autopsy. This esophagus was sectioned from above downward at seven different levels, and 345 complete sections were examined and the ganglion cells in Auerbach's plexus counted. In the narrow terminal portion of the esophagus no ganglion cells were present in 100 sections.

The chief observations made of late years by physiologists working upon the esophagus are as follows:

The Nerve Supply of the Esophagus.—Contradictory results have been obtained experimentally in animals after cutting the vagus nerve of both sides. A recent research article states that after section of both vagus nerves the lower half of the esophagus—the part supplied by the involuntary muscle—regains its tone in twenty-four hours, whereas the upper half does not

show peristalsis again for three months, and then only feebly. This observer maintains that cutting the sympathetic nerves has no added effect. In these experiments there was no dilatation of the esophagus. Another observer, whose experiments were carried out some years ago, holds that after section of the vagi the esophagus does dilate. It would seem to me that it should. The motor cells in Auerbach's plexuses are out of commission after section of the vagi unless there is a roundabout nerve supply of which we do not know.

The Cardiac Sphincter.—It will save time to admit that the musculature of the lower end of the esophagus or of the upper end of the stomach has a weak sphincter action, regardless of the presence or absence of an anatomic sphincter. Not to admit this would do violence to certain findings in experimental physiology. Cannon made the observation that when the stomach is filled with a neutral solution it runs back and forth between the esophagus and stomach without hindrance. When, however, the solution is made acid it is shut off from the esophagus and retained in the stomach. He believes, therefore, that there is an acid control of the cardiac sphincter, the nerve channel being through the cardiac plexus.

I have never been able to make out an anatomic sphincter at the cardiac end of the esophagus. In the fourteen specimens ranging in age from birth to 61 years no definite sphincter appears. A long time ago Hyrtil described an incomplete sphincter. Recently Hurst figured a very definite one at birth. My observations so far have not corroborated his. In one infant examined by sections there is a slight increase in the mass of the circular fibers at the lower end of the esophagus, but similar slight increases are found in the same specimen higher up. (See Figs. 1-5.)

We are indebted to Cannon for another important observation. He found experimentally that pressure enemia of a given portion of the intestinal tract produced experimentally was followed—four hours—by destruction of Auerbach's plexus and a cessation of peristalsis. This links up with the observation of Stokes and the later English pathologists that in cases of cardiospasm there is a loss or diminution of ganglion cells in Auerbach's plexus.

The Muscularis Mucosa.—The muscularis mucosa is thickest at birth, and thickest also at the lower end of the esophagus. From birth to adult life it increases in size only one-tenth, whereas the two outer muscular coats of the adult esophagus have increased in size to three times their measurement at birth. (McGregor.) (See Fig. 6.)

The action of the muscularis mucosa I find hard to understand. It is a longitudinal muscle and is placed a little below the epithelium. It winds in and out of the folds of the mucous membrane and is thickest where they are deepest, namely, at the terminal portion of the esophagus. This at first seems suggestive, but the axis of the folds is vertical, the same as the axis of the muscle. The muscularis mucosa, therefore, runs the wrong way to cause them. It probably reinforces the action of the longitudinal coat, the outer of the two main muscular coats of the esophagus.

The Elastic Tissue of the Esophagus.—The arteries of the body contain a large amount of elastic tissue, the presence of which brings any given vessel, for instance, the descending aorta, back to its normal size after a wave of blood has passed. The esophagus also contains elastic tissue. It is most abundant in the submucosa, although a certain amount is scattered between the bundles of the two main muscles. The elastic tissue is greatest in amount at the lower end of the esophagus. This is true of the infant as well as of the adult. Even in the disorganized esophagus of my one autopsy specimen from a case of cardiospasm the elastic tissue was more abundant at the lower end. The dog, in spite of having control of the whole length of his esophagus through voluntary muscular fibers, has more elastic tissue at the lower end than at the upper. Examination shows that the elastic tissue is scanty in the intestines. (See Figs. 7-9.)

The Comparative Anatomy of the Esophagus—This is a rather ambitious heading, because I have only a little barnyard comparative anatomy to give—that is, a description of the esophagus of the dog, the cat and the rabbit. The dog seems to stand between the cat and the rabbit, as far as its esophagus is concerned. The muscles are voluntary throughout, and are very strong. The most striking thing about the dog's esophagus is the tremendous supply of mucous glands. The dog's esopha-

gus, in consequence, must be a very greasy affair. It needs to be, considering the kind of food which he eats and his manner of eating. The characteristic ease with which a dog can vomit is due to the voluntary muscular fibers, helped out by the abundant lubrication furnished by the excessive gland supply. (See Figs. 10-12.)

In the rabbit the position of the muscular coats is reversed. The circular coat is on the outside and the longitudinal on the inside. The rabbit is said to have a cardiac sphincter. There is a slight thickening and the muscular layers interdigitate at the lower end, but I was disappointed in not finding a frank sphincter. The rabbit has few glands in the esophagus.

The Cat.—The esophagus of the cat is more like the human. It has both voluntary and involuntary muscular fibers. It has almost no glands. That the cat should be like the human is not surprising, because there are many human cats, especially female.

In the three animals just mentioned the muscular layers often run diagonally and interdigitate. In man the layers are truly circular or longitudinal.

Muscularis Mucosa.—In the esophagus of the dog there is a thick subepithelial layer of stout connective tissue. In this a muscularis mucosa of fair size is buried. No such extensive layer of subepithelial connective tissue is found in man, in the cat or in the rabbit.

The muscularis mucosa is very large in the cat, and thin in a rabbit. In the three animals under discussion the muscularis mucosa, the same as in man, is thickest at the lower end of the esophagus.

The Resting Form of the Lower End of the Esophagus.—Even if there is no anatomic sphincter at the cardia the esophagus gets along well without one. The lower end seems normally to be closed. This is brought about by the action of the elastic tissue and by the folding together of the deep vertical pleats characteristic on this part of the esophagus. In addition there is the natural twist of the esophagus on itself from right to left, and the pressure of the liver in front and the projection of the left crus into the esophagus from behind. With each inspiration the inflated lung tips press on the esophagus and momentarily close it. Keith describes the esophagus as one

long sphincter. Peristalsis is a sort of traveling sphincter. (See Figs. 13-22.)

THE PATHOLOGIC CHANGES FOUND IN CASES OF CARDIOSPASM.

The pathology which produces the main symptom of which the patient complains in cardiospasm—namely, starvation—is found in the terminal portion of the esophagus. It consists of a narrowing helped out in advanced cases by a backward bend. At the present time the English laryngologists are very active in investigating this subject. The English observers have been very fortunate in having a large number of autopsy specimens from cases of cardiospasm. For instance, Kelly, in a recent and very beautiful paper, figures eight cases collected by Owen Moore and adds three or four of his own. (See Figs. 23-27.)

A number of years ago Stokes brought out a new pathologic finding in cases of cardiospasm. He found in three esophaguses from patients who at their death suffered from cardiospasm that the motor ganglion cells which run the musculature of the esophagus and which are grouped in nerve ganglia known as Auerbach's plexuses were diminished in number. This observation was new and fundamental. When you think it over, the observation is what you would expect. When a muscle does not work, either the muscle fibers are faulty or their nerve supply is wrong. Since the publication of this observation other writers have used it as the starting point of the condition which they believe to be the cause of cardiospasm. Notably among these is Hurst. Last year, in an article in the Lancet, he mentioned that cardiospasm was caused by the tonic contraction of the cardiac sphincter due to the fact that fibrosis and infection of Auerbach's plexus had destroyed the motor ganglion cells. When Auerbach's plexus is normal, at the proper time in swallowing it sends the required stimulus to the cardiac sphincter, causing this to relax and allow food to pass. On the other hand, when the plexus is diseased, the sphincter remains tonically contracted until the weight of the food—a column eight inches high is usually sufficient—in the esophagus tires out the sphincter. All exponents of the cardiac sphincter admit that it is a weak affair.

The Findings in the Writer's Case of Cardiospasm.—The writer has had but one autopsy specimen from a case of cardio-

spasm. For him it is really a lone star case. The patient was a man in the fifties who had suffered from cardiospasm for fifteen years. He had been through the typical treatment of dilatation with bougies, under ether and without, at the hands of his physiicans and at his own hands. He would go along months or even a few years with relief and then would shut down. He was a stone cutter and finally died of tuberculosis.

His X-ray showed a much dilated esophagus, which sagged markedly to the right. It was very narrow at the lower end, and there was a pronounced backward bend at this point. The esophagus, when removed from the body, allowed water to pass into the stomach slowly and in small amounts. On pouring in water the esophagus filled to the top, ballooned out and sagged to the right, reproducing the X-ray picture. (See Figs. 23-35.)

On gross examination the dilated part of the esophagus was found to be twice as thick as the normal. Both muscular coats were of equal thickness, measuring together at their thickest portion 2.8. The thinnest portion was at the lower end, where it measured 1.9. Microscopic examination showed that both muscular layers were thickened equally. It has been stated that only the circular muscular coat was hypertrophied in these cases. By comparison with the dilated portion, the narrow terminal part looked at first glance as if it was thinner, even atrophied. On close examination this was not the case. It was of normal thickness.

Dr. McGregor has made a very exhaustive microscopic examination of this specimen.

The first finding noted was an increase of connective tissue and the round cell infiltration, and both were confined to the superficial layers of the esophagus, the mucosa and submucosa. The second observation was that the blood vessels were much increased in number and size. Both arteries and veins shared in the enlargement alike, and the enlargement suggested arteriosclerosis.

Contrary to the statement of Kelly that Auerbach's plexus is found only where involuntary muscle fiber exists—that is, from the middle of the esophagus to the end and is not found at the top—this specimen shows that the plexus is

found in small numbers at the top among the voluntary muscular fibers. (See Figs. 36-38.)

Meisner's plexus, which runs the muscularis mucosa, is an offshoot of Auerbach's plexus. As it is smooth muscle and exists throughout the esophagus, it must have a motor nerve supply. Stated broadly, however, the statement is true that Auerbach's plexus is found most abundantly at the lower part of the esophagus, where the involuntary muscular fibers predominate. Even in the dog's esophagus, where the musculature is voluntary throughout, Auerbach's plexus is found at the top.

The most important observation has to do with the number of ganglion cells in Auerbach's plexus at various levels. Sections were taken from above downward at seven different places. Auerbach's plexus was found in scanty numbers at the top of the esophagus. In the terminal portion no ganglion cells were found in one hundred sections. Fibrosis of the ganglia was not found (as reported by Hurst). The changes noted consisted of an infiltration with plasma cells in the submucosa and a disappearance of the ganglion cells. These observations, though differing somewhat in detail from the observations of Kelly and Hurst, confirm the findings of these two men and of Stokes, who made the original observation, which was to the effect that in the esophagus in cases of cardiospasm the ganglion cells in Auerbach's plexus are diminished, especially at the terminal portion.

Having found a striking increase in the number and size of the blood vessels in my cardiospasm case, I was fortunate in having the esophagus of a man of 61, who died of pneumonia after the first stage of a laryngectomy, and who showed at autopsy general arteriosclerosis of a most advanced type. His esophagus on microscopic examination showed the same condition of the blood vessels as was found in the case of cardiospasm, but what was of even more interest was the fact that the ganglion cells of Auerbach's plexus were decidedly diminished in number (McGregor). In other words, the esophagus seems to share in the general systemic disturbances which cause widespread degeneration of the organs of the body as a whole. I had not been accustomed to think of the esophagus in this light.

The observation that the ganglion cells of Auerbach's plexus are diminished or practically wanting at the terminal portion of the esophagus in long standing cases of cardiospasm is most important. But I do not think that it is the whole story. Something must keep the terminal portion of the esophagus closed when its musculature has ceased to function on account of the loss of motor nerve supply. Three things might do this, namely, pressure from the outside, the action of the elastic tissue, bend or twist. In my opinion, all three are factors. Of the three I think that bend or twist the most important.

In a typical case of cardiospasm the dilated upper portion of the esophagus is succeeded by a short narrow terminal portion. This often has a backward bend. It was conspicuous in my case. An instance of this backward bend, associated with an enlargement and an S-shaped curve of the esophagus above, has been found by the writer even at birth. In this baby the stomach had swung upward so that its long axis was horizontal. This change in the position of the stomach I believe to be a causative factor in causing the deformity of the esophagus. (See Figs. 39-41.)

While on this subject of the backward bend of the terminal portion of the esophagus, I might add that an eleventh hour bit of preparation for this paper was the gross examination and sectioning of the esophagus of a five months' fetus. On opening the chest and abdomen and exposing the esophagus for removal, it was found that the terminal portion made a U-shaped bend down and up round the left lung tip, so that the tip lay between the fundus of the stomach and the esophagus. My conjectural explanation is that the bend was due to a disproportionate growth between the lungs and the esophagus. Whether this is the right explanation or not, the fact remains, as my specimens show, that the stage may be set for trouble with the esophagus, not only at birth but long before.

The Findings in a Five Months' Fetus.—On gross examination the lower end of the esophagus, as just stated, was seen to make a U-shaped turn round the left lung tip. The upper part of the esophagus was much thinner than the lower half.

The examination of microscopic sections showed that the thinness of the upper half of the esophagus was due to the fact that the circular fibers were very sparse at the top. The

muscularis mucosa was present throughout the whole length of the esophagus. It was thin at the top and at the bottom, but relatively very thick at the middle of the esophagus. There was no evidence of an anatomic sphincter at the cardiac end.

Auerbach's plexus was found throughout the whole length of the esophagus. The number of ganglion cells to a section averaged 27, practically the same as in the infant.

THE DIAGNOSTIC BARIUM BOUGIE.

The diagnostic bougie has given me a visualization of the lower end of the esophagus which I have never had before. It has given me information which neither the X-ray nor the esophagoscope have furnished and has made me doubt some of my observations, made with the esophagoscope, at the lower end of the esophagus in years past. I have always been partial to large examining tubes. If the large tube will lead the observer astray, a small tube is a much greater sinner in this respect. The matter may be summed up as follows: It is difficult to be sure of what you see at the end of a twenty-one inch tube, large or small, and it is equally hard not to see what your mind has been made up to see. One observer sees spasm of the esophageal muscles, another spasm of the crura, another spasm of the cardiac sphincter, and finally another observer always sees stricture. (See Figs. 42-44.)

In using the diagnostic bougie, the first thing was to establish the normal dimensions of the lower end of the esophagus in its various diameters. This was accomplished by passing the bag on "Bill," the accomplished giver of enemata on the private ward of the infirmary. He was the only volunteer and will live long in my grateful memory.

At first the barium bag was used for diagnostic purposes only. It was soon found, however, that even the pressure exerted by the bag was sufficient to cause some dilatation. Then the Sippy bag, as it was stronger, with barium and cement lines, was used both for purposes of diagnosis and dilatation. Owing to the silk cover, it is possible to exert a pressure of ten to fifteen pounds. So far I have not gone beyond four pounds. I admit that watching the esophagus dilate under direct vision, which the fluoroscope allows one to do, makes one a bit timid. You feel as if the esophagus may burst at any moment.

I have had one fatality, which may have been traceable to the bag. The dilatation was without incident. Two weeks later the patient became violently ill and died with symptoms of septicemia. An abdominal exploration for the purpose of finding a rupture of the esophagus was negative. The only pathologic condition discovered was an enormously large liver, which previous X-rays showed the patient had had for some months. A small section taken from this at the operation showed a violent infection, the organism being streptococcus hemolyticus. Puncture of the liver at the time of the exploratory operation for abscess was negative. No autopsy could be obtained and so the exact condition of affairs will never be known. I am never entirely happy in passing any instrument through the esophagus, because I have had my full share of tragedies, even from the simple passing of an elastic bougie.

Findings With the Barium Bougie.—The ordinary barium picture of the esophagus in cases of cardiospasm is familiar to us all. The terminal portion comes to an awl-like point, and this point is continued as a thin ribbon of barium for an inch or an inch and a half, and then broadens out on the anterior wall of the stomach. Watched through the fluoroscope, you see the esophagus end in the same awl-like point, but at each inspiration, as the lung tips fill, the tip of the point seems to be amputated for half an inch or more. This happens regularly with each inspiration. From this picture it is hard not to draw the conclusion that the esophagus is strictured to a small caliber. On passing the diagnostic bag, however, the picture changes. After halting a second at the lower end of the esophagus the bag passes into the stomach as if it slid over a slight obstruction. On inflating the bag, the seemingly slight stricture disappears, and in its place a waistlike constriction appears at the level of the dome of the left half of the diaphragm. Under light pressure—three or four pounds—on the second or third passage of the bag, the esophagus can be seen to dilate to normal caliber. This dilatation is followed by a great improvement in the symptoms for weeks or months.

In my series of nine recent cases of cardiospasm, this waistlike narrowing at the lower end of the esophagus above the level of the crura was found in all. It dilated under light pressure, and gave the impression of a light fibrosis. The only

other explanation which I have at present for it is that it is the permanent impression of the inflated lung tips. During the life of the individual such an impression occurs eighteen times a minute, accompanied by a momentary closing of the esophagus. Some people find it hard to attach as much importance to the action of the lung tips as I do. Constant watching of their action through the fluoroscope and seeing it constantly recorded on the X-ray film have convinced me that their action must play a part in the pathology of the lower end of the esophagus. (See Figs. 45-51.)

As the barium picture is practically the same at the subsequent visits of the patient, although the barium can be seen to go into the stomach in a larger stream, and the patient reports much less difficulty in swallowing, some of the old condition which existed before the dilatation has remained or returned. The waistlike constriction is too large to obstruct of itself, so I have come to the conclusion that the return of the closure is due either to spasm at the lower end of the esophagus or to a backward twist, preferring for myself the explanation of twist. The reader, of course, can take his choice. I will only remind him that such a backward bend can be present at birth and the stage all set for trouble.

The Operative Treatment of Cardiospasm.—Mild cases of cardiospasm can be managed by dilatation, preferably dilatation by sight. Where this is unsuccessful or the case has advanced to the stage marked by bend or twist, the condition must be dealt with by some form of surgery carried out through the upper abdomen. Incision into the musculature of the lower end of the esophagus, after the fashion of the treatment of pyloric obstruction at birth, has been tried successfully in this country. (Meyer.) In England, Grey Turner has twice made a lateral anastomosis between the mobilized lower end of the esophagus and the fundus of the stomach. In the next five or ten years I feel that some such operation will be the treatment for the long standing cases, especially those with twist.

I gave the conclusions of the paper at the beginning. In closing I shall not repeat them in detail. But I should like to remind you that the terminal portion of the esopahgus in the resting position is closed. The deep vertical pleats and the abundant elastic tissue which is present at this point are the main

factors in bringing this about. The twist of the esophagus aids, so does outside pressure, especially the pressure of the lung tips. A deformity almost typical of an advanced case of cardiospasm can be present at birth. My case of cardiospasm showed a loss of the motor cells of Auerbach's plexus at the terminal portion of the esophagus, which is in line with the original observation of Stokes. Insufficient nerve supply would, of course, give altered and diminished muscular action. This must play a conspicuous part in cardiospasm, especially in the terminal stages. The esophagus is subject to degenerative diseases like arteriosclerosis. In one example of this condition the ganglion cells were found to be diminished. Important as the decrease of the ganglion cells is, and the altered muscular action resulting from this, a mechanical factor, the backward bend of the esophagus, to my mind, is equally important. It literally traps the lower end of the esophagus.

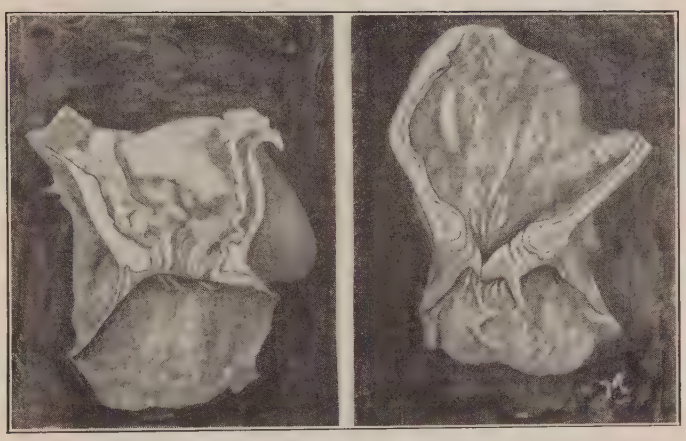


Fig. 1. Human; adult. Two specimens showing the pyloric sphincter.

Fig. 2. Retouched photomicrograph showing the pyloric sphincter. Human; adult.

Fig. 3. Baby dead at birth. The esophagus and stomach have been injected with wax. Both are normal. Notice the pyloric sphincter. See Fig. 4.

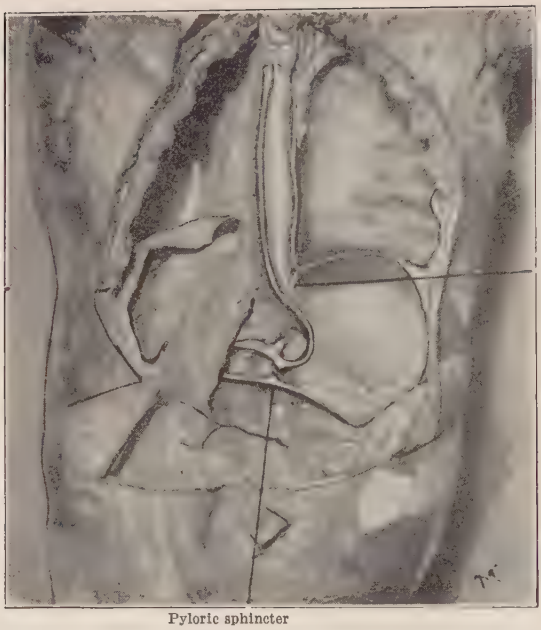


Fig. 4. Baby dead at birth. This is the same specimen shown in Fig. 3. The anterior wall of the esophagus and stomach has been removed. There is no thickening of the esophageal walls at the cardia to indicate a sphincter. There is an oblique fold on the posterior wall of the esophagus corresponding to the position of the left crus. Above this the esophagus tends to make a pocket. For many years the writer has noticed in specimens both the fold over the crus and the pocket above it. Notice the pyloric sphincter.

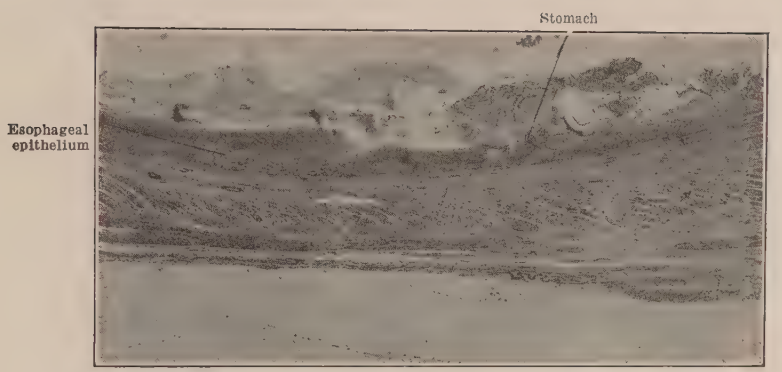


Fig. 5. Section of the lower end of the esophagus of a baby dead at birth. There is no anatomic sphincter.

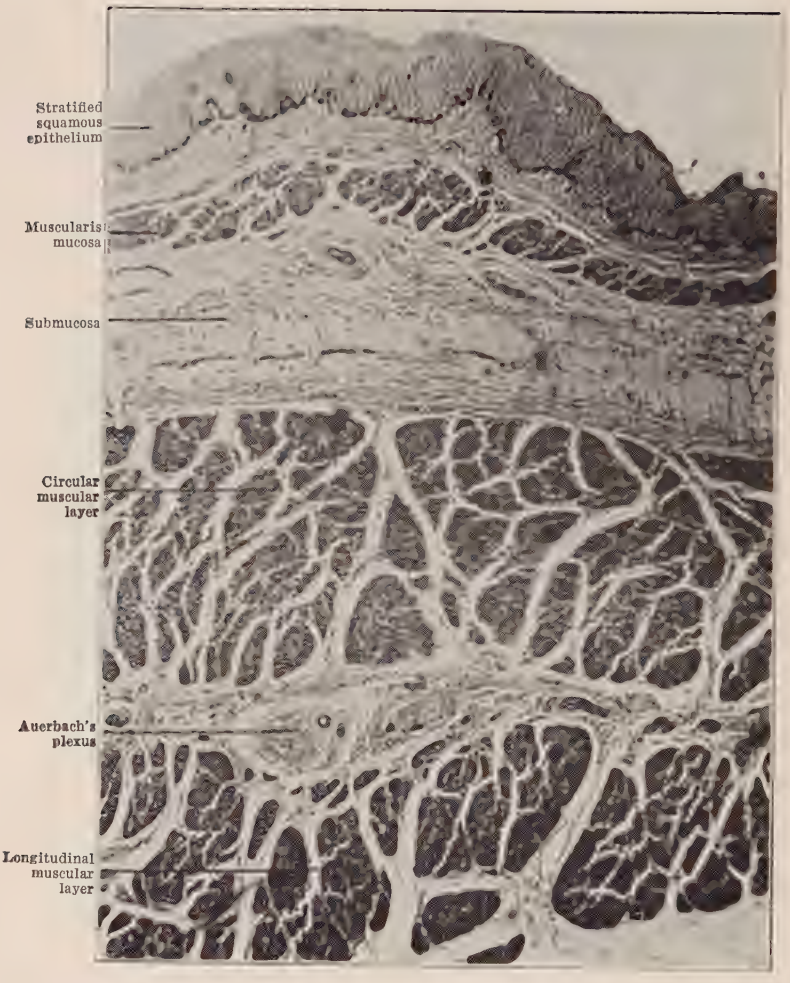


Fig. 6. Infant two years old. Section from the middle of the esophagus to show normal histology.

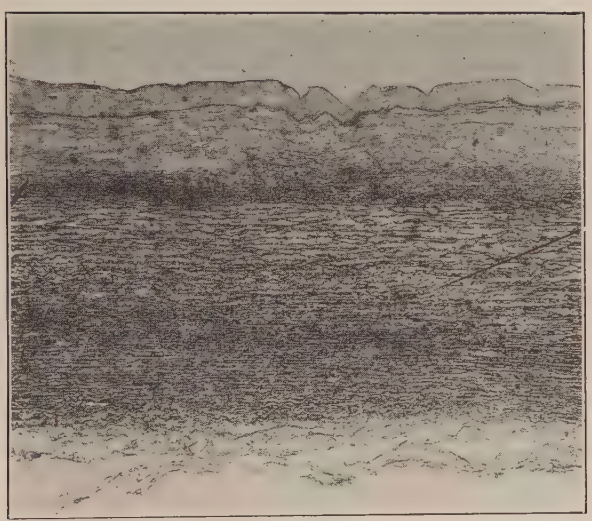


Fig. 7. Section of the descending aorta (human) stained to show elastic tissue.

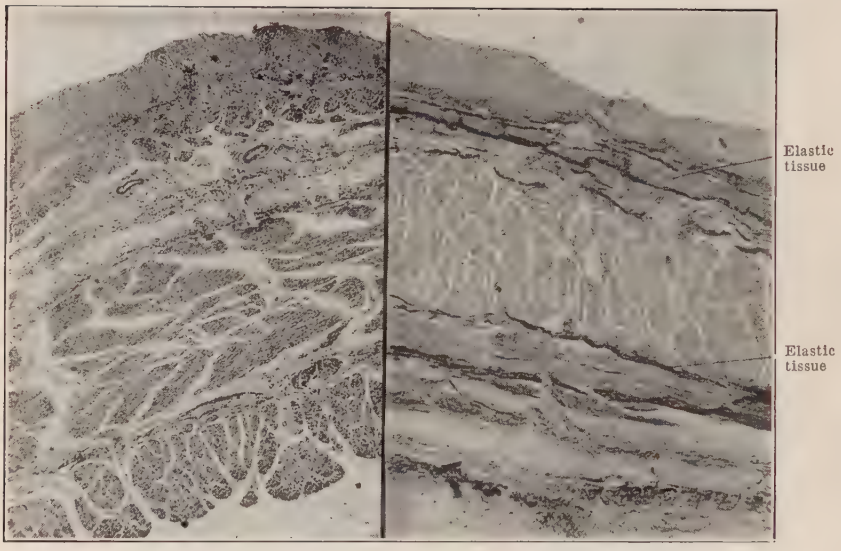


Fig. 8. Infant, two years. The first section is from the upper end of the esophagus, the second from the lower. In the first there is but little elastic tissue, in the second it is conspicuous in amount. Elastic tissue stain.

Fig. 9. Male, 61. Generalized arteriosclerosis. The lower end of the esophagus. Photomicrograph. The section shows a marked collection of elastic tissue between the submucosa and the circular layer of muscular fibres. Elastic tissue stain.

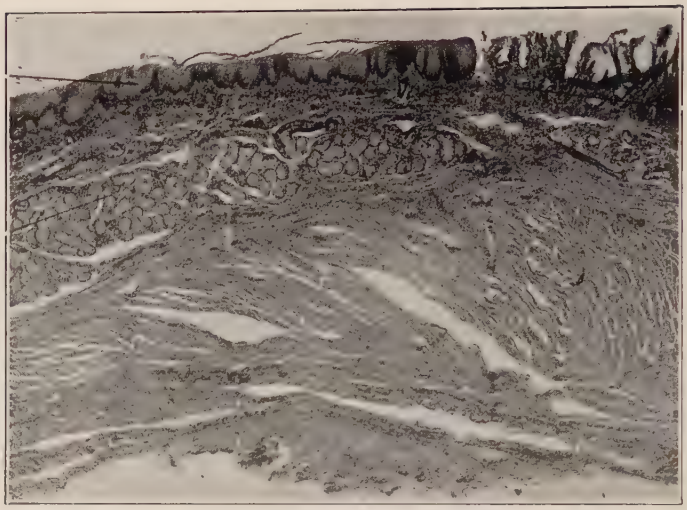


Fig. 10. Section of the lower end of the esophagus of a dog. Notice the large glands.

Fig. 11. Section of the lower end of the esophagus of a cat. Notice how abruptly the epithelium of the esophagus ends.

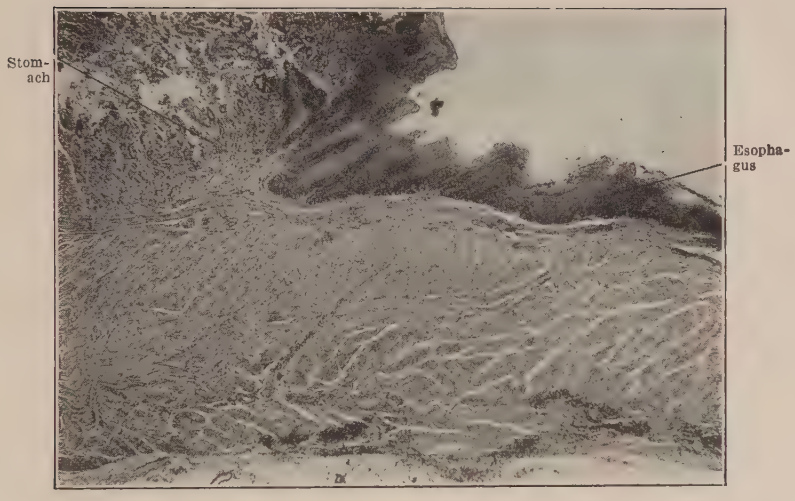


Fig. 12. Section of the lower end of the esophagus of a rabbit. The rabbit is said to have a cardiac sphincter. The writer's sections do not show one.

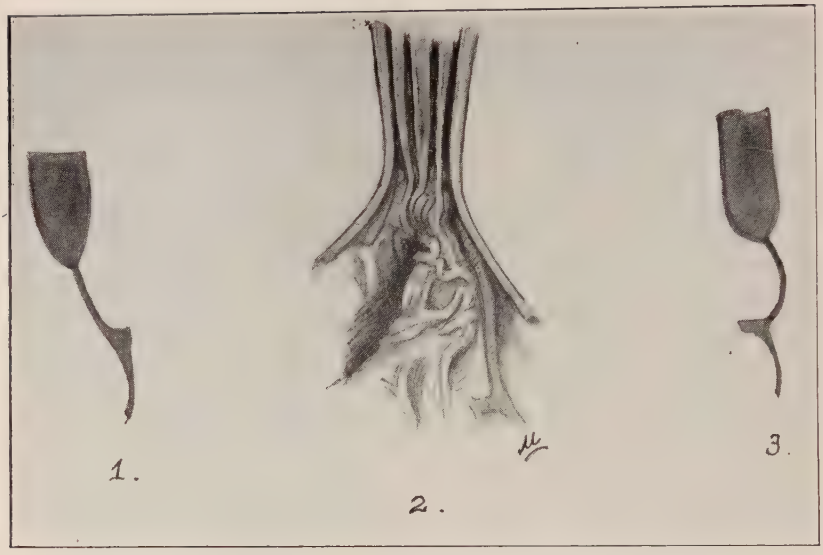


Fig. 13. The esophagus of a child. The illustration is to show the vertical pleats at the lower end of the esophagus. They are continuous for a short distance with the rougae of the stomach. On the anterior wall of the esophagus one or two pleats are continuous with long rugae which skirt the lesser curvature of the stomach. One of these rugae is shown in the illustration. In the resting or collapsed state of the terminal portion of the esophagus the barium runs in the grooves made by the pleats. This accounts for the pictures given by the barium meal. Figures 1 and 2 are such pictures. In these the esophagus is seen to come to a point. This is the point of occlusion due to the inflated lung tips. Below this there is a narrow ribbon of barium. This represents a channel between the pleats in the closed esophagus.

Where the esophagus ends and the stomach mucosa begins there is a slight ridge. Examination shows that this is due to a mounding of the mucous membrane of the esophagus. Although to the naked eye the esophageal mucous membrane seems to stop at this ridge microscopical examinations shows that it may extend fully 5 cm. below this point.

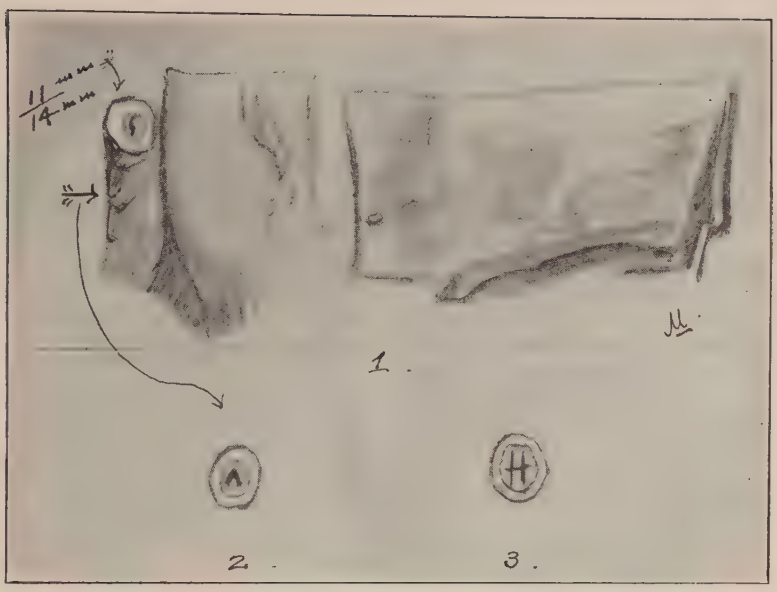


Fig. 14. Male, 61. Generalized arteriosclerosis. Specimen showing the lower portion of the esophagus and a section of the descending aorta. The second figure of the upper row is the aorta laid open to show patches of arteriosclerosis.

The esophagus in the first figure is seen to be a narrow closed tube. The closure is due to the vertical pleats. Figures 2 and 3 show the character of the pleating at lower levels. In Figure 3 that there are two deep vertical pleats, one on the anterior wall of the esophagus, the other on the posterior wall. Notice also how these two pleats practically close the esophagus. The writer believes that this is the normal resting condition of the terminal or pleated portion of the esophagus.

See Fig. 13.

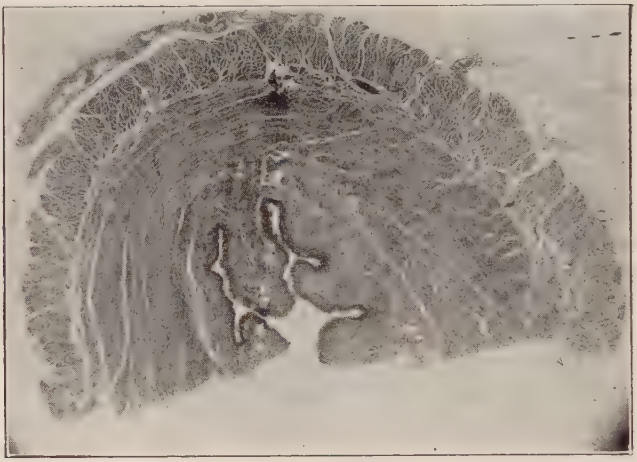


Fig. 15. Cross section of one-half of the lower end of the human adult esophagus showing the normal pleating.

Fig. 16. Male, 25. Cardiospasm one year. X-ray film of the lower end of the esophagus.

The dilated esophagus comes to the typical point. Below this there is a narrow spindle shaped ribbon—the pleated portion of the esophagus —which merges into the barium shadow along the lesser curvature of the stomach. The pleated portion in many films appears as a ribbon of uniform diameter. At times the film shows peristalsis acting at this point.

This picture with slight modifications in the shape of the pleated portion may be taken as the characteristic one obtained in cases of cardiospasm of moderate severity.

See Fig. 13.

Fig. 17. Drawing of a reconstruction of the lower end of the esophagus from frozen sections. Adult.

The drawing shows that the terminal portion of the esophagus consists of a vertical and a horizontal portion. The vertical part comes to a point and its axis changes from transverse to anteroposterior. The horizontal portion or arm is very much pleated. This part the writer believes to be closed in the resting condition. The closure is due mostly to the pleats. See Figs. 13-14-16.

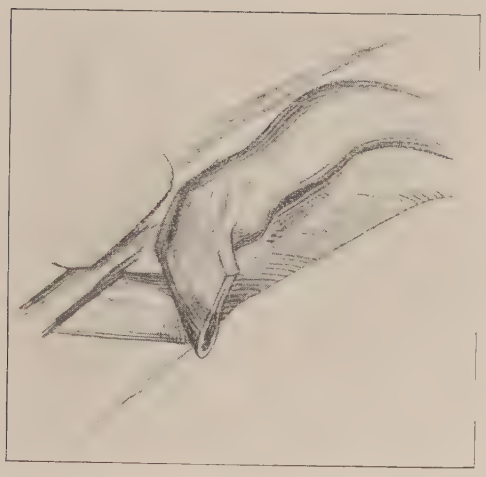
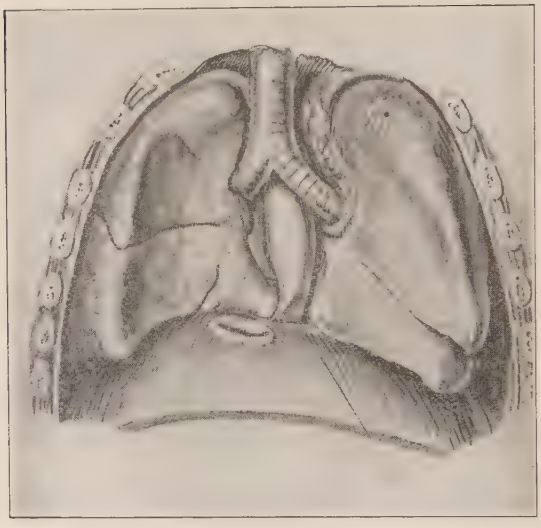


Fig. 18. Drawing of the terminal portion of the esophagus of an adult cadaver. The esophagus was not injected. The specimen shows the twist of the terminal portion of the esophagus. Notice how the left crus lies behind the horizontal arm or pleated portion of the esophagus. The anterior crus has been mostly cut away. Above the edge of the left crus there is a globular dilatation of the esophagus.



Impression of left lung tip.

Fig. 19. Drawing from a cast of the trachea, lungs and esophagus of a baby dead at birth.

The drawing shows the relation of the lung tips to the terminal portion of the esophagus. The impression of the left lung tip on the esophagus stands out clearly.

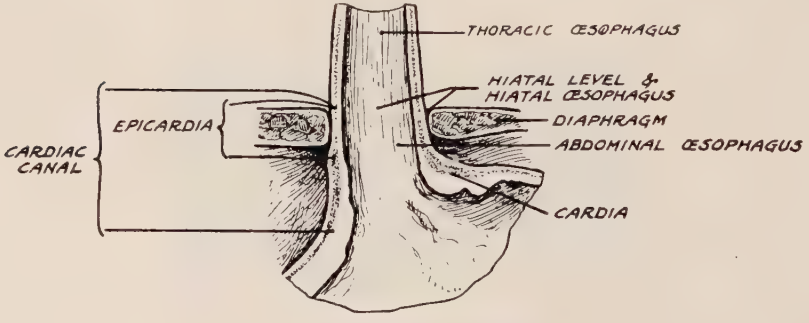
Fig. 20. Retouched tracing from X-ray film. Adult. The film was taken in full inspiration.

The film shows a very marked impression of the left lung tip on the left side of the terminal portion of the esophagus.

Fig. 21. Retouched tracing from X-ray film. The diaphragm is down and the inflated lung tips have momentarily closed the esophagus. The narrow strip extending below the closed and distended esophagus represent barium caught in the vertical folds of the esophagus.

See Figs. 13-14.

Fig. 22. Female, 40 years. Cardiospasm one year.
The barium bougie is in place. The diaphragm is down. There is a waist like constriction of the terminal portion of the esophagus above the dome of the left half of the diaphragm. The most marked portion of the constriction is on the left and corresponds to the shape of the left lung tip.



—Cardiac Canal with Names.

Fig. 23. Dissection by Professor Shattock to show the normal cardiac canal and orifice. Kelly.

Fig. 24. Female, 56 years. Dysphagia 20 years.

Autopsy specimen of the esophagus from a patient who suffered from cardiospasm. Illustration from Kelly.

Notice the dilated upper portion of the esophagus, and the narrow, tubular terminal portion.

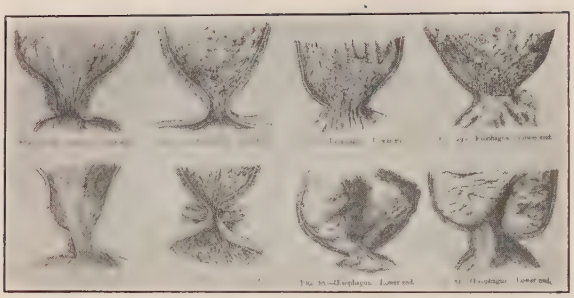


Fig. 25. Drawings of the lower end of the esophagus from autopsy specimens in eight cases of cardiospasm. The cases were reported by Moore. The figures are from Moore and are here taken from an article by Kelley who quoted them from Moore.

The first four drawings—to the middle of the illustration—show the lower end of the esophagus narrow and tubular. This tubular narrowing Moore calls the cardiac canal.

In the last four drawings there is no cardiac canal, only a fibrous ring. The stenosis of the esophagus is not as marked as in the first four cases.

The cases of cardiospasm which die of starvation are found, according to Moore, in the first group of four.

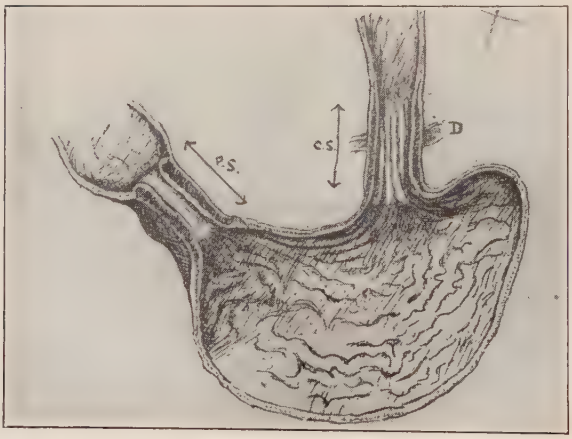


Fig. 26. Diagram from Hurst to show the cardiac and pyloric sphincters.

Fig. 27. Drawing from Hurst. Section of the esophagus from a case of cardiospasm.
The drawing shows infiltration and fibrosis of Auerbach's plexus.

Fig. 28. Retouched tracing from X-ray film. Mac D., 55; male. Cardiospasm 15 years. Death from tuberculosis of the lungs.

The film shows the typical picture of an advanced case of cardiospasm. A No. 35 French elastic bougie would pass easily.

See Figs. 29-30-31.

Fig. 29. Mac D., male; 55. Cardiospasm 15 years.
Cast of esophagus and stomach.
The esophagus is much dilated except at the lower end where there is a narrow tubular portion about an inch long. There is a marked backward bend of the esophagus above the tubular portion.

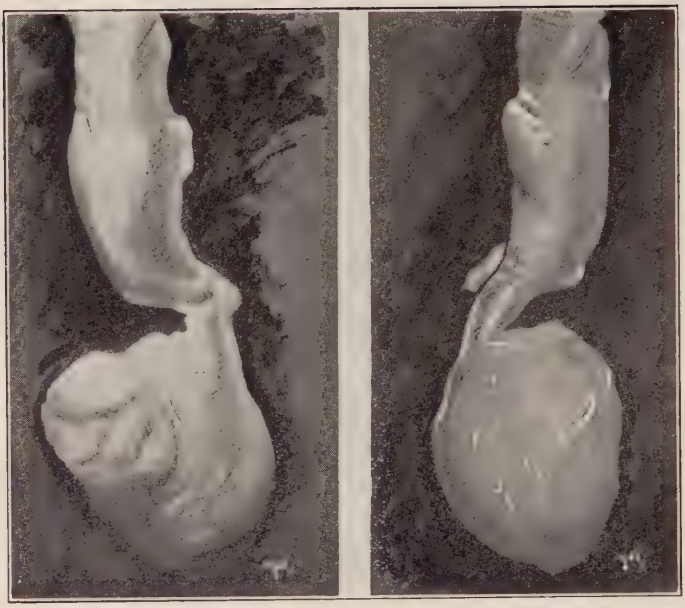


Fig. 30. Mac D., male; 55. Cardiospasm 15 years. Cast of esophagus and stomach.

The terminal portion of the esophagus seen from the right and from the left. Notice the backward bend.

The writer feels that this backward bend of the terminal portion of the esophagus plays an important part in producing the obstruction present in cases of cardiospasm. See Figs. 28-29.

Fig. 31. Mac D., male; 55 years. Cardiospasm 15 years. Death from tuberculosis of the lungs.

Photograph of esophagus and stomach. Notice the dilated upper portion of the esophagus and the narrow terminal portion.

See Figs. 28-29-30.

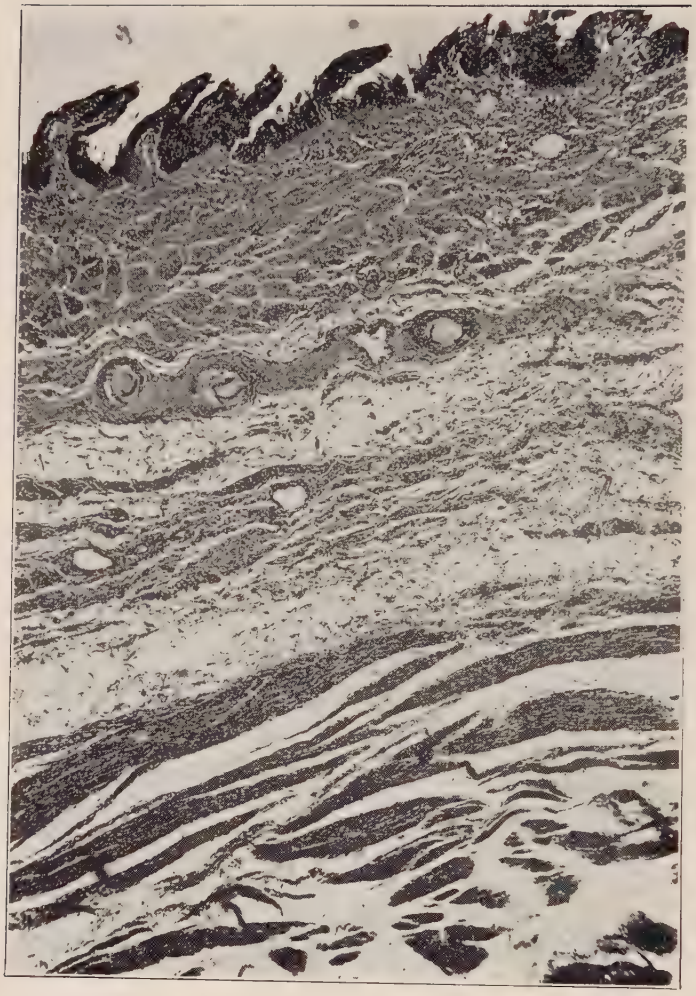


Fig. 32. Mac D., male; 55 years. Cardiospasm 15 years. Death from tuberculosis of the lungs.

Section of the esophagus showing superficial infiltration with lymphocytes and plasma cells. There is an increase of connective tissue in the infiltrated area.

Fig. 33. Mac D., male; 55 years. Cardiospasm 15 years. Death from tuberculosis of the lungs.

Section of the esophagus showing superficial infiltration with lymphocytes and plasma cells. The infiltration has penetrated the muscularis mucosa and appears beneath it round the blood vessels.

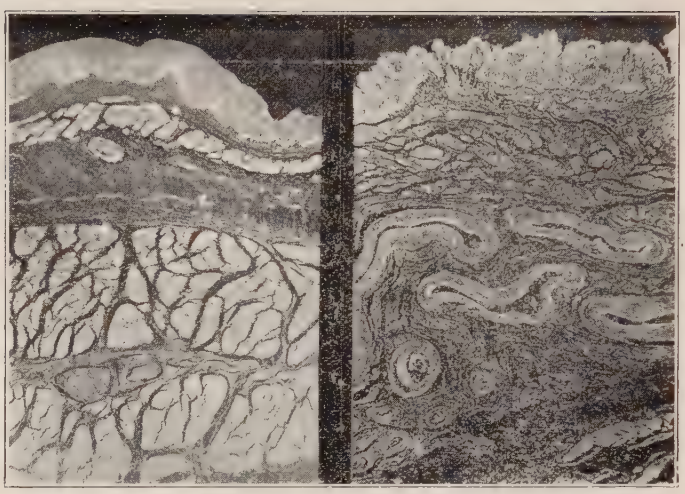


Fig. 34. The first illustration is a section of the esophagus of a child of two years. It shows but one blood vessel in the submucosa. The second illustration is a section of the esophagus from the writer's case of cardiospasm (Mac D., 55 years). Notice the increased number of blood vessels, their great size, and thick walls. Practically the same increase in the number of the blood vessels, and the same enlargement, was found in the sections of the esophagus of a man who showed at autopsy extensive generalized arteriosclerosis.

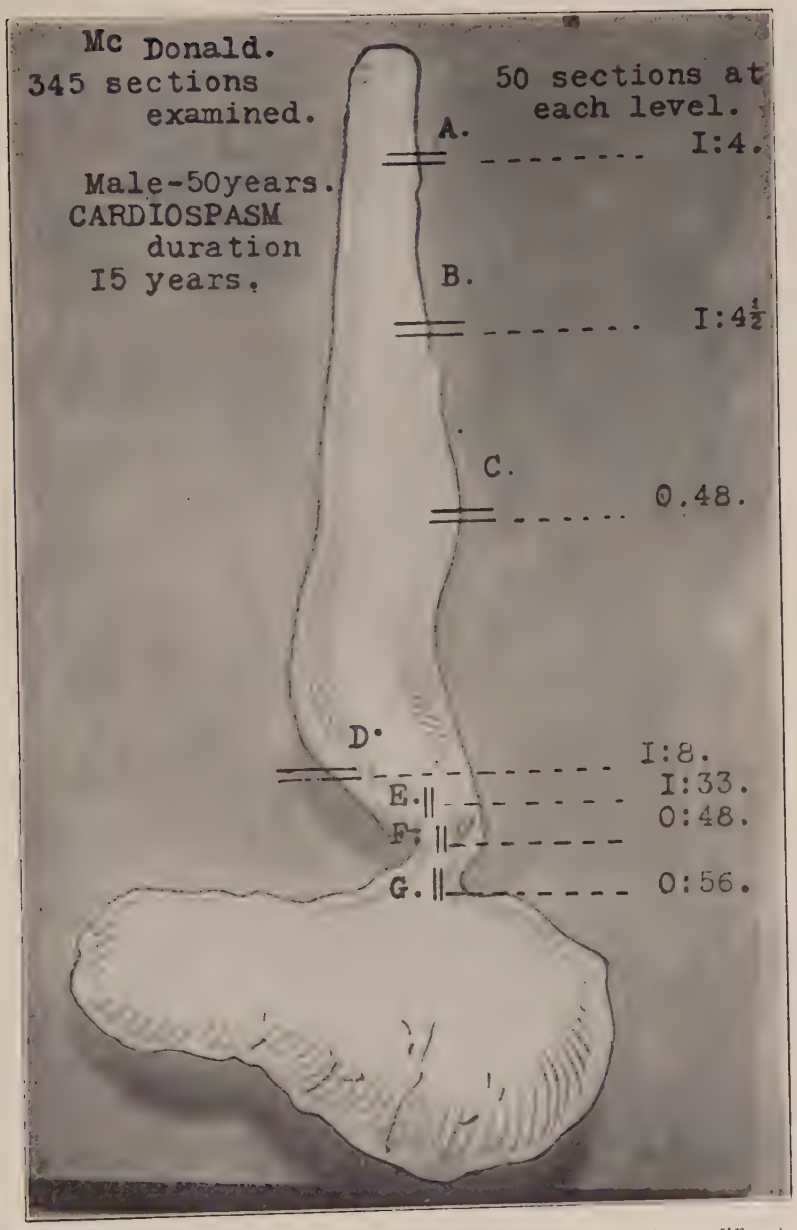


Fig. 35. Tabulation of the ganglion cell count in Auerbach's plexus at seven different levels in the writer's specimen from a case of cardiospasm. At the terminal portion of the esophagus there were no ganglion cells in one hundred sections. In an infant the average number of ganglion cells to a plexus is 27.

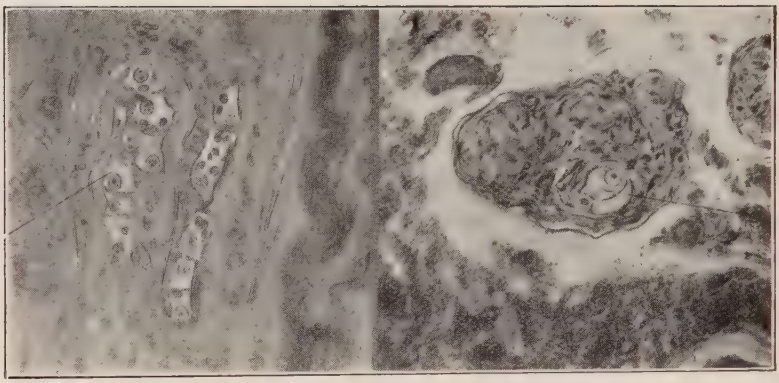


Fig. 36. Photomicrograph.

The first illustration is from the esophagus of an infant two years old. It shows one ganglion with twelve ganglion cells.

The second illustration shows an Auerbach's plexus from the writer's case of cardiospasm. But one ganglion cell remains. The reader is reminded that in this specimen at the lower end of the esophagus no ganglion cells were found in one hundred sections.

Fig. 37. Human adult, male; 50 years.

The patient had a stricture of unknown etiology at the lower end of the esophagus for ten years. He was dilated successfully under ether. Three years later a second dilatation was necessary. During this the esophagus was perforated by the bougie and he died of septic pleurisy and peritonitis.

The microscopical section shows the stricture. It is confined mostly to the region occupied by the circular layer of muscular fibres. The esophagus has lost its epithelium and the mucosa and submucosa are filled with connective tissue.

This stricture case is in marked contrast to the case of cardiospasm in which the infiltration and the increase of connective tissue was confined chiefly to the mucosa and submucosa.

See Fig. 32.

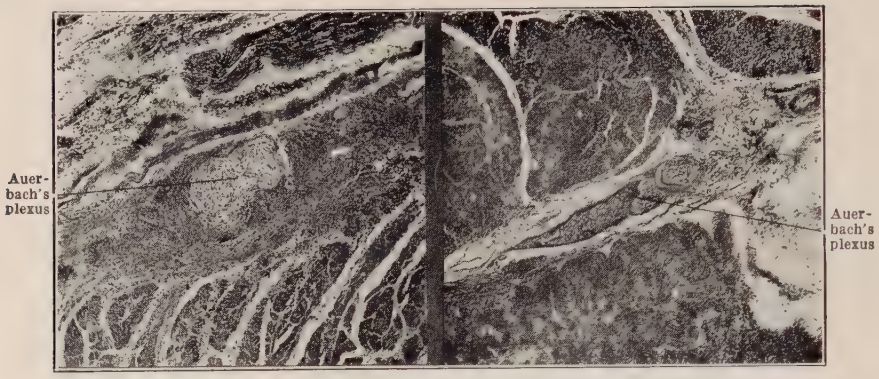


Fig. 38. Human, adult; 50 years. Photomicrograph showing Auerbach's plexus.

The patient died of septic pleurisy and peritonitis after perforation of the esophagus by a bougie.

The first illustration shows a plexus surrounded by a shower of leucocytes and plasma cells but not as yet invaded by them. In the second illustration two-thirds of the plexus has been invaded.

Fig. 39. Four views of a wax cast of the esophagus and stomach of a baby dead at birth. The stomach has swung upward until its long axis is horizontal. The esophagus is dilated, has an S-shape curve to the right, and the lower end has a pronounced backward bend.

See Fig. 41.

Fig. 40. Four babies dead at birth.

The esophagus and stomach before removal were injected with wax. The illustrations show the gradual upward swing of the stomach until its long axis becomes in the last specimen horizontal. This position of the stomach tends to produce a kink at the lower end of the esophagus.

The usual conception is that a baby's stomach is vertical. The upward swing shown in the illustrations is not, however uncommon.

Aiken

Fig. 41. Baby dead at birth.

Esophagus and stomach injected with wax. The upper part of the esophagus is flattened from before backward by the pressure of the trachea. Only the lower half is cylindrical.

The terminal portion of the esophagus bends on itself at an angle of 90° to reach the stomach.

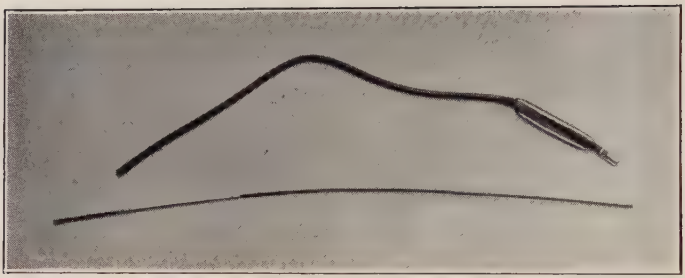


Fig. 42. The writer's diagnostic barium bougie.

The upper figure is the bougie, the lower one the whalebone staff for introducing it.

The bougie consists of a red rubber tube the size of the usual stomach tube. Fastened to the end of the tube is a blunt metal tip half an inch long and a quarter of an inch in diameter. The upper end of the metal tip has a hole bored in it to take the end of the whalebone staff and to keep it in place during the introduction of the bougie. Near the top of the metal tip there is a groove to take the thread which ties on the lower end of the rubber bag. Inside the rubber tubing at the point where the shank of the top of the rubber bag is to come there is a thin metal ring with a groove round the middle of it. This is to take the thread which ties the upper end of the rubber bag.

In the lower end of the rubber tubing at a point about under the center of the rubber bag four or five holes are punched. These allow the air which is forced into the tubing to inflate the bag. A thin rubber bag tied over the lower end of the tubing completes the main part of the apparatus. The bag is moderately inflated with air and kept distended while four barium lines are painted on it, using equal parts of barium powder and ordinary rubber cement. The lines should divide the circumference of the bag into four equal parts. They dry in twenty minutes to half an hour and will stay on the bag some six weeks. Two of the lines show in the illustration. In order to keep the lines from being washed or scrubbed off during the use of the bougie a second rubber bag is tied over the first one.

To a metal Y-tube the air bulb from a blood pressure apparatus and an air pressure gurge registering up to fifteen pounds and with a radiant dial are attached ready for coupling up with the bougie after it has been passed.

The bougie is greased and the staff also. The patient's pharynx is sprayed with a half of one per cent solution of cocaine. The patient is placed before the fluoroscope and the fluid level in the esophagus obtained by giving her—most of my patients have been women—a part of a glass of barium. A quick examination of the chest, the lower end of the esophagus, the action of the lung tips and the movement of the diaphragm is made, and then the barium which has accumulated in the esophagus is hurried on into the stomach by giving the patient a fizzing Seidlitz powder to drink. The writer hit upon this expedient for emptying the esophagus some two years ago and has since found it very much worth while. Besides emptying the esophagus and so making the passage and the retention of the bougie less messy, it gives a large gas bubble in the stomach against which the barium picture of the lower end of the esophagus stands out clearly.

The preliminary fluoroscopic examination over, the patient is seated in a chair and the diagnostic bougie is passed by touch. It usually halts a little at the lower end of the esophagus and then with a slight jump forward as if it passed an obstruction enters the stomach. If the bougie does not pass with the use of almost no force and a little manipulation the patient is placed before the fluoroscope and an attempt is made to manipulate the bougie into the stomach by sight. If the bougie does not then pass readily the attempt is given up. Usually, however, it does pass and when the patient is placed before the fluoroscope the bougie is found well down in the stomach. This being the case, the bougie is drawn up into place by sight, half of the bag being in the esophagus and half in the stomach. At this point the air pressure apparatus is connected to the bougie and the bag is inflated. So far the writer has used from four to six pounds of pressure. The thin rubber bag can be made to dilate in the stomach like a balloon. This is in striking contrast to the smaller esophagus above. While the patient is still standing before the fluoroscope four films are taken, two in the anteroposterior and two in the right lateral position. In each position one film is taken in full inspiration, and one in full expiration.

Fig. 43. In order to be able to use more pressure for dilatation than the diagnostic bougie will give, the writer uses the Sippy bag. The illustration shows the bag with the air pressure apparatus attached.

Four barium lines are painted on the bag and the silk cover drawn on. In order to take away the harshness of the corners of the silk cover a rubber bag is tied in place over it. The silk cover keeps the Sippy bag from dilating unduly. This apparatus is capable of exerting a pressure of thirteen to fifteen pounds.

The Sippy bag can be used for diagnostic purposes as well as for dilating. The thin bag of the diagnostic bougie, however, as it balloons so readily in the stomach, gives a more dramatic picture.

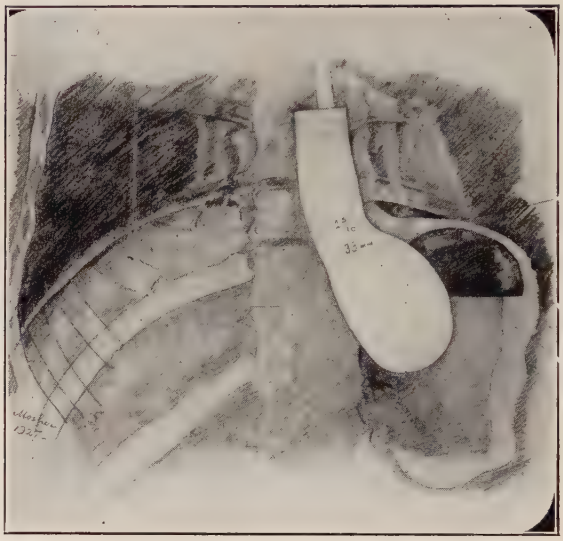


Fig. 44. Adult, male; 30 years. Normal esophagus. Retouched tracing from X-ray film. The diagnostic barium bougie is shown in place. It gives the normal transverse diameter of the terminal portion of the esophagus to be 33 mm. The anteroposterior diameter is practically the same.

In this case when the diaphragm was down there was but a slight narrowing of the esophagus.

Fig. 45. Male, 30 years. Cardiospasm one year. The upper four figures are the barium meal findings, the lower four the findings with the barium bougie. Figs. 1-2, 4-5 are films taken in the anteroposterior position. Figs. 3-4, 7-8 are from films taken in the lateral position. Figs. 2-4-6-7 were taken with the diaphragm down.

The films taken with the barium meal give the impression that the esophagus is tightly strictured. The lower four figures from films of the barium bougie in place show that this is not the case.

Notice the impression of the left lung tip shown in Figs. 6-7-8.

Notice also how the terminal portion of the esophagus—the pleated portion—is practically closed.

See Figs. 2-4 of this plate.

Fig. 46. Female, 40 years. Cardiospasm one year.
Lateral film. The diagnostic barium bougie is in place. It shows a long waist like constriction of the terminal portion of the esophagus.

Fig. 47. Retouched tracings from X-ray films of four cases of cardiospasm. Figs. 1-2 are from the same patient. So are Figs. 5-6.

The films show the variations in the size of the waist like constrictions shown by the barium bougie. In these cases the esophagus dilated to normal size with a pressure not exceeding four pounds in three. The one which did not is No. 4 in the series.

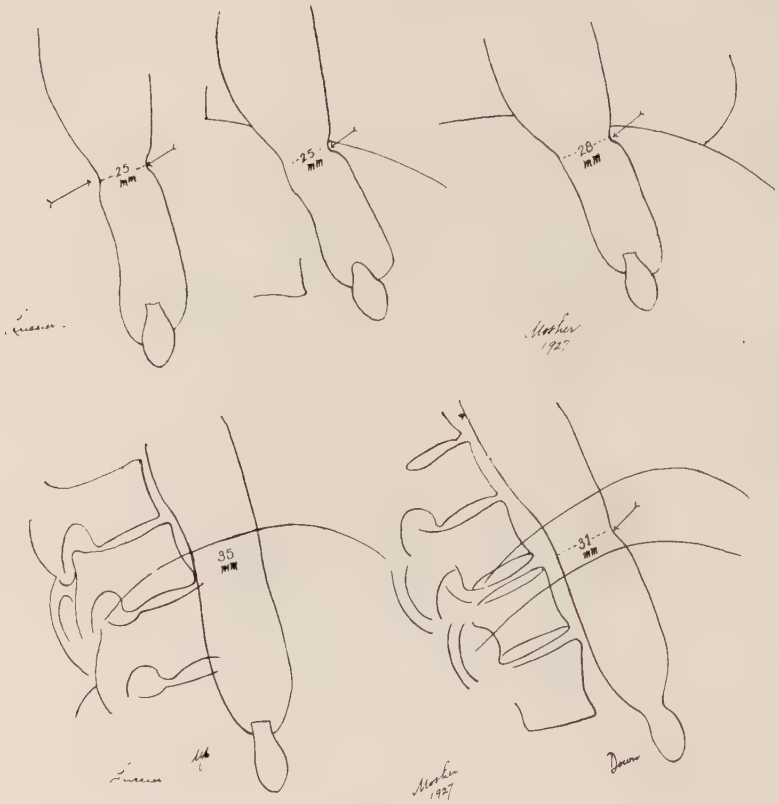


Fig. 48. Female, 35. Cardiospasm nine years.

Tracings from X-ray films showing the findings of the barium bougie. The first three films were taken in the anteroposterior position and the last two in the right lateral.

Notice how in the fourth figure the esophagus has dilated to the normal diameter (33mm).

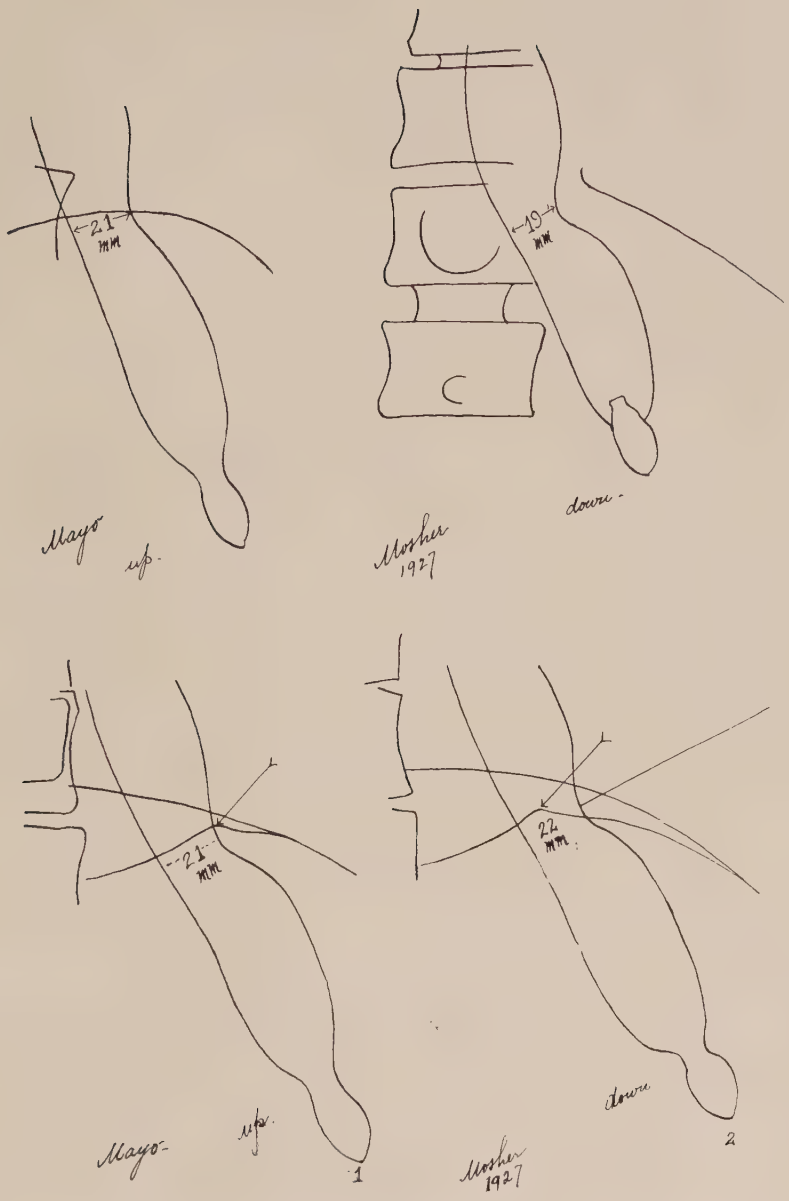


Fig. 49. Male, 32. Cardiospasn four years.

Tracings from X-ray films showing the findings of the barium bougie. The first two films were taken in the anteroposterior position, the second two in the lateral position.

Notice in the second figure the marked impression of the left lung tip. In the third and fourth figures the diaphragm shows a pronounced adhesion indicating previous pathology in the chest.

This case had a gastrostomy and retrograde bouginage. It is one of the two cases which have not dilated to normal.

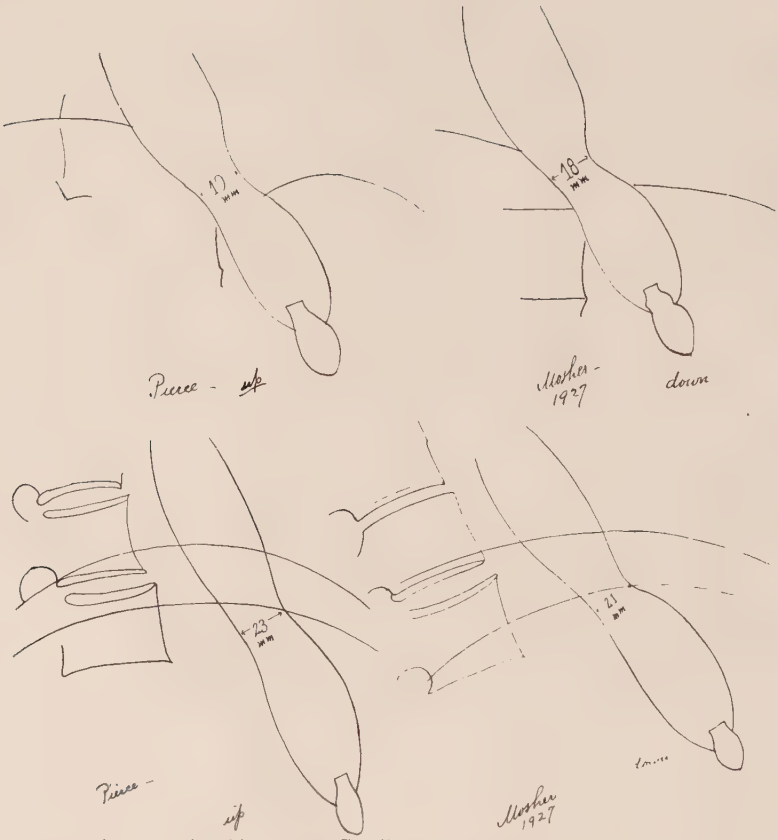


Fig. 50. Female, 45 years. Cardiospasm two years.

Tracings from X-ray films showing the findings of the diagnostic bougie. The first two figures are from films taken in the anteroposterior position and the second two from films taken in the lateral position.

This patient has been dilated twice and the esophagus so far has not dilated to normal. This case is one of two in a series of nine which has not dilated to normal.

Notice in the second figure the marked impression of the left lung tip. See Fig. 49.

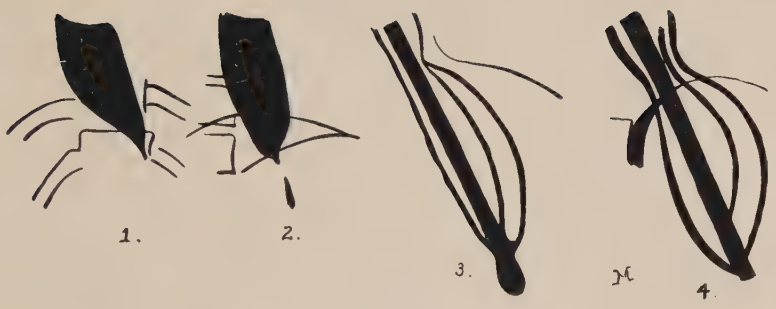


Fig. 51. Female, 20 years. Cardiospasm one year.

Figs. 1-2 are the barium milk findings.

Figs. 3-4 are the diagnostic bougie findings.

Figs. 1-3 are anteroposterior films; 2-4 lateral films.

Notice in 3 the very marked notch on the left wall of the esophagus. This is opposite the left lung tip.

In 4 the narrowing is seen to extend—waist like—all round the esophagus. It is more marked in front than behind.

These contrasted illustrations show the added information furnished by the diagnostic bougie over the barium meal.

16.F.53.
A study of the lower end of the 1928
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